Management of retinal detachment: a guide for non-ophthalmologists

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Patients with retinal detachment often present to their general practitioner, emergency department, or optometrist after central vision has been compromised. This delay is unfortunate because early repair results in little or no visual loss. Once the detachment extends across the fovea (the central macula), permanent visual impairment is almost inevitable. Thorough examination of the retina needs equipment that is rarely available to non-ophthalmologists. Recognition of symptoms and awareness of the risk factors for retinal detachment may help in making speedy referrals and saving vision.

What is retinal detachment?

Retinal detachment is separation of the neurosensory retina from the underlying retinal pigment epithelium (fig 1). In health, the potential "subretinal space" between these two layers is closed by the retinal pigment epithelium actively pumping fluid across the retina and into the choroid.¹ Cellular interdigitation and extracellular matrix provide additional adhesion. Retinal detachment occurs when the forces of retinal attachment are overcome and fluid accumulates in the subretinal space.

How common is retinal detachment and why should it interest non-ophthalmologists?

Large, population based studies of retinal detachment find an annual incidence of around 1 in 10 000, and a familial aggregation study estimated a lifetime risk of 3% at age 85.^{2,3} White and Asian populations have similar rates, with a lower incidence among blacks.^{4,5} The average age of presentation is around 60, with the sexes affected equally.²

The modest incidence belies the importance of retinal detachment as a true ophthalmic emergency. Although most detachments can be repaired surgically, only those treated early avoid permanent visual impairment.⁶ Many patients first present to general practitioners, emergency departments, and optometrists. Retinal detachment is therefore of clinical (and possibly medicolegal) interest to non-ophthalmologists.⁷

Pathogenesis of retinal detachment

Retinal detachments can be caused by scarring of the vitreous and retina (tractional) or leakage of fluid into the subretinal space (exudative), but most follow the development of breaks in the retina (box 1), which allows fluid in the vitreous cavity to enter the subretinal space (fig 2). Such detachments are called rhegmatogenous (from the Greek word *rhegma*, meaning a break, rent, or fissure).

Most retinal breaks form when the vitreous separates from the retina as part of the normal ageing process. This event, posterior vitreous detachment, is the result of a lifetime process of degenerative liquefaction and shrinkage of the vitreous (fig 2).⁸ Although posterior vitreous detachment is benign in most people and may go unnoticed, those with symptoms carry a 10-15% risk of developing retinal breaks.^{9,10} Posterior vitreous detachment is rare before the age of 40, but the prevalence increases steadily thereafter, to around 40% in the seventh decade. By the ninth decade, up to 86% of the population develop a partial or complete posterior vitreous detachment.¹¹

What symptoms should alert me to a threatened retinal detachment?

Flashes and floaters

Photopsia and floaters can occur in conditions other than posterior vitreous detachment (box 2). Photopsia associated with posterior vitreous detachment results from traction on the retina as the vitreous pulls away. It is usually described as recurrent, brief flashes in the

Methods

For an overview in the current management of retinal detachment we consulted textbooks and proceedings of meetings in the field of retinal surgery. We searched Medline and Cochrane Review databases for "retinal detachment", "retinal break", "retinal tear", "retinal hole", "vitreous detachment", "lattice", "retinal tuft", "vitrectomy", "scleral buckle", and "retinopexy", and we searched the internet for reviews and perspectives on retinal detachment.

Fig 1 | The retina lines the internal surface of the posterior two thirds of the globe. It is thickest around the optic nerve and ends at the ora serrata, 5-7 mm behind the limbus. The macula lies temporal to the optic nerve, bordered by the vascular arcades; the fovea is a depression at its centre that provides fine visual acuity. The outermost layer of the retina contains photoreceptors (rods and cones), loosely attached to the retinal pigment epithelium; they depend on the retinal pigment epithelium and choroid for support. The vitreous completely fills the vitreous cavity and is firmly attached to the retina near the ora serrata, over the optic nerve and macula, along the blood vessels, and around degenerative retinal lesions

temporal peripheral field, but can occur anywhere. Floaters are caused by vitreous opacities casting shadows on the retina. Posterior vitreous detachment makes them more mobile and thus more noticeable. Vitreous condensation around the optic nerve often manifests as an irregular ring or crescent shaped opacity ("Weiss ring") after posterior vitreous detachment (fig 2). Some patients recall a dramatic event of bright flashes accompanied by showers of black dots that later coalesced into "strands," "cobwebs," or "cloudy haze." Such descriptions suggest vitreous haemorrhage from avulsed blood vessels (fig 2) or the liberation of retinal pigment epithelial cells through retinal breaks.

Symptomatic posterior vitreous detachment carries a considerable risk of breaks that are likely to progress to retinal detachment. Autopsy studies have shown that 4-9% of the population will develop asymptomatic retinal breaks in their lifetime, most of which do not progress to detachment.^{8 12} In contrast, a retrospective case series of 295 patients presenting with photopsia or floaters found retinal detachment in 61% of 80 eyes that had developed retinal breaks.¹³

Visual field defects, blurring, and distortion

When the retina is separated from the retinal pigment epithelium, the visual field defect is opposite the site of the detachment because of the optical inversion of

images. It is commonly described as a dark curtain or shadow, appearing first in the periphery and moving to the centre over hours, days, or even weeks as the detachment extends. Visual acuity decreases when the macula becomes detached, and the patient may notice distortion of images. Without prompt treatment, total retinal detachment and blindness are almost inevitable.

Who is at risk of developing retinal detachment?

Retinal detachment occurs more commonly with age as posterior vitreous detachment becomes more prevalent. Cataract surgery is thought to accelerate vitreous liquefaction and posterior vitreous detachment.¹⁴ A retrospective, population based study found that the eight year cumulative risk of retinal detachment approached 1% after cataract surgery, almost nine times higher than expected.¹⁵

Box 1 Types of retinal detachment

Rhegmatogenous

Caused by breaks in the retina

Associated with:

Age

Myopia

Cataract surgery

Trauma

Degenerative retinal lesions

Stickler's syndrome

Juvenile X-linked retinoschisis

Marfan's syndrome

Tractional

Caused by chronic traction from scars on the retinal surface and across the vitreous cavity

Associated with:

Proliferative diabetic retinopathy

Proliferative vitreoretinopathy

Retinopathy of prematurity

Penetrating eye injury

Sickle cell retinopathy

Retinal vein occlusion

Exudative

Caused by leakage of fluid into the subretinal space

Associated conditions:

Inflammatory (uveitis, scleritis)

Hydrostatic (malignant hypertension, toxemia of pregnancy)

Neoplastic (choroidal melanoma, haemangioma, metastasis)

Vascular (Coat's disease, retinal macroaneurysm)

Maculopathy (neovascular macular degeneration, central serous chorioidoretinopathy)

Congenital disorders (nanophthalmos, optic disc pit)

Fig 2 | Rhegmatogenous retinal detachment. Hyaluronic acid in the vitreous holds water and keeps insoluble collagen fibrils dispersed in the gel matrix. A—with aging, changes to hyaluronic acid cause pockets of liquefied vitreous, leaving the collagen fibrils to condense into larger fibre bundles, which appear as chronic floaters. B—pockets of liquid vitreous coalesce to form larger spaces. Defects in the vitreous cortex let liquid into the plane between the vitreous cortex and retina, initiating posterior vitreous detachment. C—the collapsing vitreous exerts mechanical traction on the retina and optic nerve, which may be perceived as flashing lights; condensation of the vitreous around the optic nerve may appear as a crescent shaped floater (Weiss ring). Vitreous traction may lead to avulsion of blood vessels or formation of retinal breaks. D—fluid enters the subretinal space through the retinal break and retinal detachment develops

The risk increases further if vitreous is lost during surgery.¹⁶

Myopic patients (with increased axial length) are more likely to develop posterior vitreous detachment at a younger age.¹¹ The peripheral retina in these eyes is less robust and commonly harbours degenerative lesions, such as lattice, where the retina is thinned and firmly adherent to the vitreous.¹⁷ Retinal detachment from atrophic holes, without posterior vitreous detachment, is relatively common in highly myopic people.¹⁸

Fig 3 | Management of retinal detachment

Cataract surgery in very myopic patients carries a particularly high risk of detachment.¹⁹

Many retrospective studies have shown that trauma is an important cause of retinal detachment in young patients.^{20 21} A direct blow to the eye induces breaks in the retina, usually in the form of retinal dialysis (lifting of the retinal edge at the periphery), and tears and atrophic holes from retinal contusion can also develop. Ocular trauma induces premature posterior vitreous detachment, possibly through liquefaction of the vitreous from leakage of blood and protein.²²

Prospective and retrospective case series report retinal detachment in up to 23% of second eyes as the features that played a role in the previous detachment are replicated.^{23 24} Family history is also a risk factor because features such as increased axial length and degenerative retinal lesions are heritable traits.²⁵

How to assess a patient with suspected retinal detachment

If the presenting symptoms and risk factor profile suggest retinal breaks or detachment, further ophthalmic assessment is indicated. The completeness of the assessment will depend on the availability of equipment and the skills of the examiner (fig 3).

Visual acuity should be assessed before the pupil is dilated. Decreased visual acuity usually indicates macular detachment, but vitreous haemorrhage may also reduce vision. A confrontational visual field test may show an asymptomatic peripheral field defect. Extensive retinal detachment will produce a relative afferent pupillary defect. This can be tested by shining a bright torch alternately on one eye for two seconds, then rapidly swinging it on to the other eye. This can be tested by shining a bright torch on each eye for two seconds, several times in quick succession. The pupils will dilate when the torch is swung on to the affected eye

Box 2 Causes of photopsia and floaters

Photopsia (perception of light not attributable to an incident light)

Posterior vitreous detachment
Flick phosphene
Migraine
Postural hypotension
Choroidal tumours
Optic nerve pathology
Transient ischaemic attacks

Floaters (perception of mobile spots, lines, or haze due to vitreous opacities)

Age related macular degeneration
Posterior vitreous detachment
Vitreous haemorrhage (diabetic retinopathy, trauma)
Asteroides hyalosis
Uveitis
Retinitis pigmentosa

Fig 4 | Funduscopy appearance of rhegmatogenous retinal detachment. The patient noticed blurred vision in her left eye three days earlier. A sector of retina is attached superiorly; shallow retinal detachment over the macula and nasally appears pale and featureless owing to the masking of the choroidal pattern. The fovea appears dark against the pallor of detached macula, and the bullous retinal detachment inferiorly appears pale, opaque, and wrinkled. The detachment was caused by a single superotemporal retinal tear

and constrict when it shines on the unaffected eye, as a result of less “brightness signal” being sent to the brain from the eye with the detachment.

Dilating the pupil with a short acting mydriatic (tropicamide 1%, for example) is safe, and the risk of inducing acute angle closure glaucoma is extremely low. The red reflex should be examined with a direct ophthalmoscope at 1 metre for loss caused by retinal detachment or vitreous haemorrhage. If a detachment is near the macula it may be visible through the direct

Fig 5 | Surgery for retinal detachment. A—in scleral buckle surgery, the retinal break is treated with cryotherapy or laser therapy, and an explant (usually a silicone band or strip) is sutured on the outer surface of the sclera to indent the wall of the globe. This interrupts the flow of fluid through the break, allowing it to close. Subretinal fluid is drained through a small sclerotomy or left to be absorbed into the choroid. B—the vitrectomy approach involves removing the vitreous through sclerotomies made in the pars plana. Subretinal fluid is drained internally, and laser therapy or cryotherapy is applied around the flattened retinal break. The vitreous cavity is filled with a tamponade (usually gas but occasionally silicone oil) to hold the retina in place while scarring develops around the break. In some cases, pneumatic retinopexy may be less invasive: a bubble of gas is injected into the vitreous cavity, and the patient’s head is positioned to place the bubble on the retinal break; once the retina is flattened, the break can be treated with laser therapy or cryotherapy

ADDITIONAL EDUCATIONAL RESOURCES

Review articles

Brucker AJ, Hopkins TB. Retinal detachment surgery: the latest in current management. *Retina* 2006;26(suppl 6):S28-33.

Gariano RF, Kim CH. Evaluation and management of suspected retinal detachment. *Am Fam Physician* 2004;69:1691-8.

Ghazi NG, Green WR. Pathology and pathogenesis of retinal detachment. *Eye* 2002;16:411-21.

Scott JD. Future perspectives in primary retinal detachment repair. *Eye* 2002;16:349-52.

Internet resources

Emedicine—www.emedicine.com/emerg/topic504.htm (Larkin GL. Retinal detachment. April 2008.)

Handbook of Ocular Disease Management—www.revoptom.com/handbook/sect5r.htm (Sowka JW, Gurwood AS, Kabat AG. Retinal detachment. 2001.)

ophthalmoscope; the detached retina will appear pale, opaque, and wrinkled, with masking of the underlying choroidal pattern (fig 4).

Retinal detachment cannot be excluded by direct ophthalmoscopy owing to the narrow field of view. Slit lamp or indirect ophthalmoscopy with a condensing lens is needed to examine the peripheral retina and locate retinal breaks. A slit lamp is needed to assess the anterior vitreous for pigment granules (“tobacco dust”), which correlate with a 90% risk of retinal breaks.²⁶ The anterior vitreous is best visualised with an oblique slit beam through a dilated pupil.

Macroscopic vitreous haemorrhage is associated with a 70% risk of retinal breaks.²⁷ When dense vitreous haemorrhage precludes examining the fundus, ultrasonography can identify the detachment. Because ultrasound does not image the retinal periphery well, retinal breaks there cannot be diagnosed with certainty by this method.

ONGOING RESEARCH

The scleral buckling versus primary vitrectomy in rhegmatogenous retinal detachment study (SPR study) aims to determine the best approach for managing more complex retinal detachments

Sutureless vitrectomy systems promise less invasive surgery and reduced discomfort

Pharmacological vitreolysis may simplify repairs of difficult tractional detachment

Tissue adhesives designed to seal retinal breaks may obviate the need for intraocular tamponade

Research continues in ways to prevent proliferative vitreoretinopathy, which remains the leading cause of failed surgery

Decisions to refer: when, to whom, and how urgently?

All patients with a recent onset of retinal detachment should be referred immediately. Time can be saved by referring the patient directly to the ophthalmologist who will perform the surgery. In some countries, retinal detachments are mostly repaired by specialist vitreoretinal surgeons. If immediate referral is not possible, the patient should be instructed to lie down with the face on the side of the detachment to the pillow (opposite the field defect) to minimise the detachment extending towards the macula.

There is no general consensus on how soon patients presenting with a symptomatic posterior vitreous detachment and no other visual symptoms should be referred for a definitive examination. The referral should be made as soon as possible, certainly within days, in view of the considerable risk of retinal breaks associated with the presentation. The patient should be instructed to return earlier if symptoms worsen or a visual field defect develops.

Treatment of retinal break and detachment

Retinal breaks caused by posterior vitreous detachment are treated using laser therapy or cryotherapy to create a scar adhesion between the retina and retinal pigment epithelium. This treatment is almost 100% successful, but new breaks can develop elsewhere.²⁸ Prophylactic treatment of asymptomatic breaks or degenerative retinal lesions has not been shown to reduce the risk of retinal detachment.²⁹

Once the retina is detached, additional surgical procedures are required to reattach it and seal the breaks. Most retinal detachments not involving the macula are repaired on the same day or the following day. For patients presenting with the macula already detached, the macula should be reattached within five days, but the urgency of the surgery is influenced by individual factors such as the duration of symptoms, the height of macular detachment, and visual acuity.⁶

Scleral buckling and vitrectomy with gas tamponade are the most common surgical approaches to repairing retinal detachment (fig 5). Pneumatic retinopexy can be performed in some cases, but enthusiasm for this technique outside North America has been minimal. In 95% of cases the retina will be reattached, sometimes after more than one operation.³⁰ Successful

SUMMARY POINTS

Retinal detachment affects 1 in 10 000 people each year, but the incidence is much higher in association with myopia, cataract surgery, trauma, previous retinal detachment, and family history

Most retinal detachments can be repaired successfully, but the key to optimum visual recovery is prompt diagnosis and treatment

Photopsia and new floaters are symptoms of posterior vitreous detachment, which precedes retinal detachment

Retinal detachment should be considered in any patient presenting with an acute onset of visual symptoms, and these patients should be referred urgently

If symptoms are accompanied by decreased vision or visual field loss, referral should be immediate

reattachment of the retina does not always correlate with a good visual outcome, and patients presenting with poorer vision are less likely to achieve good final visual acuity.⁶ Macular detachment has a poorer visual outcome, and the chance of regaining good vision diminishes with the duration of detachment.⁶ Results are best when the detachment is repaired before the macula becomes involved, and this can be achieved only through early diagnosis and urgent referral.

Postoperative care

After the operation, topical antibiotics and corticosteroids are routinely prescribed, and cycloplegics and ocular hypotensive agents may be prescribed in some patients. If intraocular gas has been instilled, vision will be poor. As the gas is resorbed over weeks to months, the gas-fluid interface will become apparent to the patient as an undulating line that moves downward. Worsening vision is not expected and should be reported to the surgeon immediately. Severe pain is also unusual and should be reported. Headache and nausea suggest an acute rise in intraocular pressure. Patients with intraocular gas are usually asked to maintain a certain head posture, typically for one week. Air travel must be avoided while the gas remains, and intraocular gas is a contraindication for volatile gas anaesthesia.

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INFORMATION RESOURCES FOR PATIENTS

All About Vision—www.allaboutvision.com/conditions/retinadetach.htm

Health Encyclopedia—www.healthscout.com/ency/68/207/main.html

Macula Center—www.maculacenter.com/EyeConditions/RetinalDetachment.htm

Medicine Net—www.medicinenet.com/retinal_detachment/article.htm

Medline Plus—www.nlm.nih.gov/medlineplus/ency/article/001027.htm

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On the nose

While on holiday in France from my work in Thailand, I thought I had a left upper molar infection. I wished I'd stocked up on some antibiotics (far too easy to purchase in resource poor settings, where they can least afford problems with drug resistance). The next day I diagnosed left maxillary sinusitis. The following day the supposed sinusitis was now on the left side of my nose as a 1.5 cm, red, slightly raised, circular lesion.

In Thailand superficial migrating skin lesions are caused by helminths. The commonest is cutaneous larva migrans, where humans are accidental hosts for the cat and dog hookworm. Strongyloides cause larva currens from autoinfection of larvae penetrating the perianal skin. Gnathostomiasis is acquired by eating uncooked foods such as fish, shellfish, frog, or chicken. My lesion was not the classic, erythematous, tunnel-like lesions of cutaneous larva migrans as shown in textbooks. Skin lesions from strongyloides tend to be fleeting and itchy, which mine wasn't, whereas gnathostomiasis tends to present as migratory skin swellings or subcutaneous lesions.

I went to the local pharmacy to buy some anthelmintics—albendazole or topical thiabendazole. "C'est impossible"—no easy, over the counter options for wormy things in France. Welcome back to Western medicine. Later that day a courteous and concerned French general practitioner with an enthusiasm for blood tests checked me for eosinophilia, C reactive protein, and, to top it off, *Gnathostoma spinigerum* antigen.

I waited a few days for the results of raised eosinophils and slightly raised C reactive protein ... and a few months for the antigen result, which was negative. It turned out that the lab in France had sent the sample to Thailand for testing; the result went back to France, by which time I was back in Thailand.

Meanwhile, I wasn't allowed anthelmintics in France because I was lactating—whereas our bamboo clinic in Thailand uses mebendazole and albendazole routinely for women in their second and third trimesters and postpartum if they have positive stool results or migrating superficial skin lesions, in line with the WHO Millennium Development Goals.¹ So I was unable to access a basic drug in a resource rich country (France) when we dispensed it liberally in our resource poor setting and it was available over the counter in my native Australia.

Back at the nose, things were getting uncomfortable as the helminth wormed its way to the bridge: there's not a lot of space for objects under the skin in that area. I turned to our household cure-all, tea tree oil, soaked on to a dressing on my nose while I slept. In the morning there was no sign of further movement, and within 48 hours the lesion was completely gone. After this palaver, I looked up the medicinal properties of the oil (*Melaleuca alternifolia*): it can be useful for various skin infections,² but this is the first report I know of treating a superficial helminth infection.

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- 2 Carson CF, Hammer KA, Riley TV. *Melaleuca alternifolia* (tea tree) oil: a review of antimicrobial and other medicinal properties. *Clin Microbiol Rev* 2006;19(1):50-62.